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The Effect of Intravenous Catheter Diameter on the Temperature of Fluids Warmed by the Level 1™ Fluid Warmer

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This is to certify that the thesis prepared by Timothy F. Bruce entitled **The Effect of Intravenous Catheter Diameter on the Temperature of Fluids Warmed by the Level 1™ Fluid Warmer** has been approved by his committee as satisfactory completion of the thesis requirement for the degree of Master of Science.

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THE EFFECT OF INTRAVENOUS CATHETER DIAMETER ON THE TEMPERATURE OF FLUIDS WARMED BY THE LEVEL 1™ FLUID WARMER

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Nurse Anesthesia at Virginia Commonwealth University

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ABSTRACT

The Effect of Intravenous Catheter Diameter on the Temperature of Fluids Warmed by the Level 1™ Fluid Warmer

Timothy F. Bruce, BSN

Medical College of Virginia--Virginia Commonwealth University, 1990

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A quasi-experimental design was chosen to determine what effect intravenous catheter diameter had on the temperature of fluid warmed by a rapid infusion warmer (i.e., Level 1™ Fluid Warmer). The study was conducted in an operating room under simulated surgical conditions. One bag of Ringer's lactate solution was infused by gravity (108 cm height) through the Level 1™ Fluid Warmer and then through each of 4 different sizes of intravenous catheters (i.e., 18 ga, 16 ga, 14 ga, and 8.5 Fr). Temperature was measured in degrees Celsius at the following sites: (a) the operating room, (b) the fluid bag, (c) the exit point of the warmer, (d) the exit point of the air eliminator, and (e) the hub of the intravenous catheter. The difference in fluid temperature between the exit point

of the warmer and hub of the intravenous catheter was used to determine heat loss. Room temperature was maintained at 21.0 ± 0.5^{0} C. Results of the study indicated that catheter diameter had a significant effect on fluid temperature. While no heat loss occurred with the 8.5 Fr intravenous catheter, fluid temperature progressively decreased with each smaller catheter size. Data analyses were performed using a one-way analysis of variance.

Chapter One

Introduction

Hypothermia, a body core temperature of 35 degrees Celsius (^{0} C) or less, is common in anesthetized patients and often associated with numerous detrimental side effects (Martyn, 1981). Various causes exist for the development of hypothermia and many are intrinsic to the surgical environment. Cool operating room temperatures, cold skin preparation solutions, and cold intravenous fluids are some of the prevalent factors contributing to the hypothermic state. Recognizing these preexisting conditions, anesthetists usually employ multiple means to prevent hypothermia in the surgical patient.

These preventive methods incorporate either external or internal strategies which combat heat loss from the patient. Some external forms of warming include the use of (a) heated blankets, (b) plastic coverings, (c) higher ambient operating room temperatures, and (d) warmed skin preparation solutions. Two major internal warming mechanisms include the administration of warmed intravenous

products and humidification of inhaled gases. The former internal methodology is crucial to normothermic maintenance when large amounts of fluids are rapidly infused as in trauma or other conditions with larger perioperative hemorrhage.

Extensive fluid replacement requires efficient heating systems that can both warm and infuse fluids quickly. Newly developed rapid infusion warmers heat and deliver fluids much faster than their conventional predecessors. Recent technology allows massive transfusions of intravenous fluids at suitable temperatures to conserve body heat. However, because these advanced warming devices were designed specifically for infusion rates up to 1,000 milliliters per minute (ml/min), their performance may be impaired at vastly slower rates (Flancbaum, Trooskin & Pedersen, 1989).

Slower infusion rates may result from the use of small diameter intravenous catheters. The Hagan-Poiseuille law governing fluid flow states that the principle determinant of resistance to flow is the radius of the tube (Barker & Tremper, 1989). Utilizing the Hagan-Poiseuille equation, it is possible to determine that a 16-gauge (ga), intravenous catheter (1.7 millimeters [mm] diameter) has more than 9 times

the resistance of a 14 ga, intravenous catheter (2.1 mm diameter). Therefore, assuming all other variables remain constant, fluid infusion capability decreases with smaller diameter catheters.

Since catheter size directly affects the rate of flow, it may also indirectly affect fluid temperature. As flow rate decreases with smaller diameter catheters, the time of fluid exposure to cool ambient air increases. At rates considerably less than 500 ml/min, significant fluid temperature reductions are known to occur as fluid travels from the warmer to the patient (Russell, 1969). It seems logical then to assume that even more heat loss would occur from fluids passing through decrementally smaller intravenous catheters. Therefore, using a rapid infusion warmer, this study examined the amount of heat loss occurring with each of the four most popular sizes of intravenous catheters.

Statement of the Problem

Fluid warmers are used to combat hypothermia by heating intravenous fluids prior to patient administration. Recently developed rapid infusion warmers heat fluid more readily, and infuse it faster than conventional fluid warmers (i.e., warm water bath devices). However, the diameter of intravenous

catheter ultimately determines the exact rate of fluid flow. Since slower infusion rates (< 20 ml/min) cause heat loss from fluids warmed by conventional warmers (Baker, 1985), similar loss may occur using rapid infusion warmers with smaller intravenous catheters.

Statement of the Purpose

Research on conventional fluid warmers proves heat loss occurs proportional to flow rate (i.e., slower rates result in greater loss) (Baker, 1985; Russell, 1969). The purpose of this study was to determine if a statistically significant loss of heat occurs using a rapid infusion warmer at flow rates limited by intravenous catheter size.

Hypothesis

There is no difference in fluid temperature from a rapid infusion warmer at flow rates determined by varying intravenous catheter diameters.

Variables

Independent. The independent variables include (a) the intravenous fluids warmed by the Level 1 Fluid Warmer, and (b) the intravenous catheter diameter.

Dependent. The dependent variable is the difference in fluid temperature measured between the exit point of the warmer and the entry point of the patient.

Theoretical Definitions

The following theortecial definitions are employed in this study:

Temperature. The amount of heat possessed by matter. It was measured and expressed numerically in degrees Celsius (^{0}C) for this study.

Heat loss. The difference in measured temperature between two points. For purposes of this study, these points included the exit point of the warmer and the hub of the intravenous catheter.

Rapid infusion fluid warmer. A device capable of warming intravenous fluids to a minimum of 35^0 C at flow rates less than 1,000 ml/min.

Flow rate. The amount of fluid delivered in a specific time frame and expressed in ml/min.

Operational Definitions

The following operational definition was used in this study:

Intravenous catheter. A hollow plastic cannula inserted intravenously allowing the delivery of fluid and ranging in size from 18 ga to an 8.5 french (Fr) diameter.

Assumptions

1. The temperature probes and thermometers were accurately reflecting temperatures within manufacturers' specifications.

2. The timing device correctly measured infusion rates for each sized intravenous catheter.

Limitations

1. More than one electronic temperature device was used in this study.

2. The room temperature might have varied during the time of the experiment.

Delimitations

1. All thermometers used self-calibrating mechanisms.

2. Thermostatic control of room temperature was maintained at 20.0 degrees Celsius ($\pm$ 0.5^0 C).

Conceptual Framework

Thermal homeostasis. Humans are warm blooded organisms known as homeotherms. Like other mammals, man maintains a remarkably constant internal temperature of 37^0 C, even though the environmental temperature can vary widely (Tortoral & Anagnostakos, 1978). Thermal regulation is both voluntary and involuntary. While seeking shelter from the cold is obviously a voluntary action, perspiring in warm weather is distinctly an involuntary or autonomic form of temperature control.

The body's ability to regulate body temperature originates from the hypothalamus. Located in the uppermost part of the brain stem, the hypothalamus senses internal and external body temperatures, and responds accordingly. It initiates heat production if core temperature drops, but quickly dissipates heat when temperature rises (Hall, 1978).

Heat conserving and relieving mechanisms are usually quite reliable at maintaining adequate body temperature. While shivering and vasoconstriction help to conserve heat, dissipation of heat occurs through dilation of cutaneous blood vessels and via perspiration. All of these activities require intact central neuronal coordination and normal neuromuscular

function (Hall, 1978). Because anesthesia can seriously impair these networks, anesthetized patients usually have compromised thermoregulatory systems.

Mechanisms of heat loss. The four different mechanisms by which the body loses heat include (a) conduction, (b) convection, (c) evaporation, and (d) radiation. Each one can rob the body of vital warmth. Depending on the degree and number of methods involved, dramatic heat loss is possible. While in the operating room, the surgical patient encounters all four means of heat reduction.

Conductive heat loss occurs by the transfer of body heat via direct contact with a cooler surface. The patient loses heat by lying on a cool operating table. Heat is also lost when the surgical drapes or other cool object touches the patient's skin (Barker & Tremper, 1989).

Another form of heat loss is convection. As cool air contacts the body, it becomes warmed and leaves by normal current flow (Tortora & Anagnostakos, 1978). The faster the air moves, the more rapid the heat loss. Since a standard operating room is ventilated with 10 to 15 room air changes per hour, convection is a constant threat to the patient's thermal status (Barker & Tremper, 1989).

The surgical patient also loses heat due to evaporation. Liquids contacting the skin, especially preparation solutions, cause additional heat loss through evaporation. About 0.58 kilocalorie is needed to evaporate each gram of water from the skin. Therefore, the evaporation of only 150 milliliters of water is sufficient to remove all the heat produced by the body during basal conditions (Tortora & Anagnostakos, 1978). Because basal metabolic rate decreases with anesthesia, the surgical patient is at an even greater risk for evaporative heat loss.

Finally, body heat is lost by radiation. Similar to conduction, it involves the transfer of heat to other objects. However, no physical contact with this cooler object occurs. Instead, heat is absorbed by walls, ceilings, floors, and equipment in the operating room that have cool surface temperatures. Heat is radiated from the patient to these cooler surfaces. Even if the air is warmed, loss by radiation continues until all material in the environment is warmed to a temperature equal to that of the patient (Barker & Tremper, 1989).

Hypothermia. A body temperature of 35^0 C or less, as measured by a rectal, an esophageal, or a tympanic monitor is considered hypothermia (Martyn, 1981). It

is further classified according to the degree of severity. Mild, moderate, and severe categories exist to describe the various physiological effects that occur with increasing stages of hypothermia.

In mild hypothermia (core temperature between 33^0 and 35^0 C), shivering, peripheral vasoconstriction, and tachycardia are common (Lonning, Skulberg & Abyholm, 1986). Vasoconstriction helps to insulate the body, while shivering produces heat by muscle contraction. Tachycardia increases cardiac output which replenishes muscle energy demands. Mild hypothermia also causes urinary diuresis leading to sluggish circulation due to dehydration and elevated hematocrit (Felicetta, 1979).

During moderate hypothermia, core temperature is between 30^0 and 33^0 C. Consciousness is frequently lost due to the depressant effect of cold on the central nervous system (Andersen, 1973). Shivering decreases, and stiffness of muscle and joints is common (Lonning et al., 1986). Cardiac output decreases secondary to reduced tissue metabolism and increased vascular resistance. Metabolic reduction is also responsible for a decrease in respiratory frequency (Jessen & Hagelsten, 1972). Numerous pathological cardiac conduction changes accompany moderate hypothermia and may persist for days after rewarming.

In the most serious state, severe hypothermia, body temperature drops below 30^0 C. Many of the pathophysiological conditions seen in moderate hypothermia remain, but are greatly magnified (Lonning et al., 1986). For example, as temperature progressively falls, cardiac arrhythmias range from atrial fibrillation to asystole. Initially, hypothermia shifts the oxyhemoglobin dissociation curve to the left, resulting in less oxygen dissociating from hemoglobin. Subsequently, less oxygen is delivered to the tissues. Because these patients are extremely hypothermic, they develop severe acidosis. This acidotic condition shifts the oxyhemoglobin curve back to the right, thus enhancing oxygen delivery (Marcus & Edwards, 1978). Unfortunately, any significant benefit from this change in the oxyhemoglobin curve is probably negated by the overall reduction in cellular perfusion. Another serious consequence of severe hypothermia is a two or even three-fold decrease in most enzymatic processes (Gunby, 1980). The resulting hypoglycemia from exhaustion of glycogen stores can devastate an already compromised physiological status.

<u>Pathophysiology of hypothermia</u>. Hypothermia affects the entire body. The physiological abnormalites that occur are based on the duration and

intensity of the hypothermic state. Therefore, to fully understand the results of decreased body temperature, it is important to examine how each organ system responds.

Hypothermia causes injury to cells in the human body by one or more of the following mechanisms: (a) decreased cellular metabolism; (b) slowed membrane transport, (c) cell dehydration, resulting in toxic levels of enzymes and electrolytes; (d) damage from intracellular water freezing; and, (e) anoxia due to less oxygen released to cells by hemoglobin (Martyn, 1981). The extent of cellular damage depends upon the degree of hypothermia sustained, while the consequences that result are based on the location and function of the cells involved. Because brain and nerve cells cannot regenerate, hypothermic damage to these areas is extremely serious.

Initially, hypothermia increases blood pressure due to peripheral vasoconstriction. As temperature continues to fall, both bradycardia and myocardial depression develop. The resulting decrease in cardiac output causes a reduction in blood pressure from its early rise (Lilly, 1986).

The fall in cardiac output follows the local cooling of the pacemaker cells and the Purkinje

conduction system (Martyn, 1981). The electrocardiogram (EKG) reflects these conduction changes. For example, cooling of the heart increases the length of the PT-QS and PR-QT intervals (Martyn, 1981). Another frequent EKG abnormality seen with hypothermia is the Osborn J-wave, an acute elevation of the ST segment resulting from the temeprature's depressant effect on the sino-atrial note (Elder, 1984). Arrhythmias accompanying these conduction changes occur at specific body temperatures.

Ventricular irritability occurs at 30^0 C followed by ventricular fibrillation at 28^0 C. If the body temperature drops further, cardiac arrest is probable. Attempts to warm the patient after the arrest are dangerous because of the acid load returning to the heart from the periphery. The increased load results in an increased oxygen demand and puts the heart at risk for ischemic injury (Lilly, 1986).

As the respiratory control center in the medulla cools from hypothermia, the breathing rate decreases (Martyn, 1981). This relationship causes an increased retention of carbon dioxide leading to respiratory acidosis. At 24^0 C, spontaneous respiration usually ceases (Martyn, 1981).

Depression of normal enzymatic activity by cold causes reductions in the metabolic rate (Elder, 1984). As a result, both oxygen consumption and carbon dioxide production decrease. However, hypoxia and respiratory acidosis are common with hypothermic states. While decreased ventilatory function contributes to each of these conditions, hypoxia occurs mainly due to a leftward shift in the oxyhemoglobin dissociation curve (Lilly, 1986).

The oxyhemoglobin dissociation curve describes how hemoglobin delivers oxygen to the tissues and the factors that influence its ability to do so. When the curve shifts to the left, oxygen is more tightly bound to hemoglobin and is less available to the tissues. Because hypothermia causes a left shift of the curve, tissue hypoxia commonly results.

Decreased body temperatures impair the kidney's ability to concentrate or dilute urine. As temperature falls, tubular transport of sodium, chloride and water progressively decrease (Rupp & Severinghaus, 1986). Additionally, tubular reabsorption of ions is limited, causing greater excretion of potassium (Rupp & Severinghaus, 1986). Hypothermia produces vasoconstriction which may be interpreted as fluid overload (Lilly, 1986). This suppresses the release of

antidiuretic hormone and "cold diuresis" occurs resulting in dehydration, agglutination, and sludging of blood in renal vessels (Martyn, 1981). Eventually, cardiac output decreases, causing tubular ischemia and oliguria.

In hypothermia, the liver blood supply decreases due to reduced cardiac output. As a result the liver's ability to conjugate and detoxify the blood becomes seriously depressed (Lonning et al., 1986). The slowing of these enzymatic pathways delays the metabolism and clearance of many anesthetic drugs.

Blood clotting ability is also diminished by the hypothermic liver. At temperatures of 35^0 C and below, platelets are sequestered in the portal circulation producing thrombocytopenia and impaired coagulation (Lilly, 1986). These coagulopathies increase the potential for hemorrhage and serious blood loss in the hypothermic patient.

For each 1^0 C reduction in body temperature, cerebral blood flow decreases 6 to 7%. Also, cerebral metabolic rate for oxygen and glucose decreases 7 to 10% (Elder, 1984). This reduced demand for oxygen and glucose is most responsible for the cerebral protection afforded by hypothermia (Rupp & Severinghaus, 1986).

Cooling of the central nervous system may produce dullness. When the temperature of the brain falls between 32^{0} and 30^{0} C, loss of consciousness occurs (Andersen, 1973). As extremities cool, impairment in the conduction of peripheral nerve impulses causes muscular dysfunction (Vanggaard, 1975). Hence, reflexes are usually absent in severely hypothermic patients.

Hypothermia and anesthesia. Simply stated, hypothermia affects the administration and the outcome of anesthesia. Slowed metabolism of drugs, delayed awakening, and increased postoperative oxygen consumption secondary to shivering are only a few of the problems encountered with hypothermic patients. The anesthetist's understanding of the anesthetic implications of hypothermia aid in the provision of safe anesthesia.

One important consideration of intraoperative hypothermia is its potentiating effect on inhalational anesthetics. Hypothermia reduces the minimum alveolar concentration (MAC) for these agents because of its own depressant effects on the central nervous system (Vitez, White & Eger, 1974). Therefore, a less concentrated dose is needed to achieve a satisfactory level of anesthesia. Recovery from inhalational

anesthetics also requires additional time due to decreased volatile drug metabolism and hypothermic neurological depression (Lilly, 1986).

A second consequence of hypothermia is prolonged neuromuscular blockade with certain muscle relaxants. Conflicting data exists concerning the exact mechanism of this enhancement. However, research shows that the hypothermic state has different effects on the following: (a) nerve axonal conduction velocity, (b) the rate of acetylcholine release, metabolism, and resynthesis, (c) the muscle membrane system, and (d) muscle relaxant drug metabolism and distribution (Elder, 1984).

Postoperatively, the most severe consequences of hypothermia are shivering and hypoxia (Hall, 1978). The muscle activity associated with severe shivering can increase oxygen consumption by 500%. If ventilation and cardiac output are decreased by surgery and residual anesthesia, venous blood becomes desaturated (Hall, 1978). This desaturation, combined with depressed ventilatory function, causes arterial hypoxia.

Another problem caused by hypothermia in the recovery period is postoperative hypertension resulting from vasoconstriction (Hall, 1978). Cardiac output is

suppressed by this hypertensive state reducing oxygen supply below demand levels. Furthermore, when vasodilation occurs with rewarming, it may reveal significant fluid deficits previously hidden by the hypertension.

Causes of hypothermia. Multiple environmental and physiological factors contribute to the development of hypothermia. Heat loss in the surgical patient results from the combined effect of surgery, anesthesia, and perioperative maintenance. The physical condition of the patient also helps determine the risks and consequences of the hypothermic episode.

Regarding the surgical risk of hypothermia, the type and extent of surgery performed relates to the amount of heat loss expected. Patients having large surgical wounds (e.g., abdominal or thoracic), and those undergoing trauma surgery or major vascular procedures are susceptible to serious losses of body heat (Lilly, 1986). Additionally, operations exposing large body surfaces to cool ambient air for long time periods can cause significant hypothermia by convection, radiation, and evaporation.

Anesthesia may also jeopardize the patient's normothermic status. Thermoregulation of the patient is compromised both centrally and peripherally by

anesthetics (Hall, 1978). Vasodilation can occur by epidural and spinal anesthesia along with inhalational agents such as halothane. Heat loss results from blood cooling in the dilated skin vessels. The use of neuromuscular blocking agents contribute further to hypothermia by abolishing the shivering reflex. Additionally, the inhalation of cold, dry anesthetic gases reduces core temperature in a dose and time dependent manner.

Intravenous fluid administration is another major cause of heat loss in surgical patients. The infusion of large quantities of fluids at room temperature causes a reduction in total body heat until such fluids are warmed to body temperature (Hall, 1978). The heating of these products by the body consumes nearly all of the patient's caloric production. This caloric expenditure decreases temperature rapidly, especially in trauma cases where massive transfusions are common. Along with trauma patients, certain other patients are equally at risk for developing hypothermia. Young children and elderly adults are more prone to become hypothermic due to particular physiological attributes. Neonates depend on both brown fat metabolism and physical activity for heat production. Both these mechanisms are severely limited by anesthesia. Small

children also have large body surface areas relative to volume and scant subcutaneous fat for insulation, which allow for greater heat loss. In elderly adults, heat producing metabolism is reduced and there is less muscle mass to produce heat by shivering. Most importantly, any increase in oxygen demand for heat production is extremely hazardous due to elderly patients' decreased cardiovascular reserve (Hall, 1978).

Prevention of hypothermia. Preventing hypothermia in the surgical patient is a prime goal of the anesthetist. The selection of one or more of the various techniques designed to maintain normothermia can include (a) warming the operating room to at least 23 to 24^0 C, (b) using infrared heat lamps, (c) placing warming blankets under patients, (d) covering the patient as much as possible with cotton blankets, mylar-aluminum foil blankets, or plastic wraps, (e) draping the head, (f) heating and humidifying anesthetic gases, and (g) warming intravenous fluid and blood. The anesthetist often combines several of these methodologies, which can significantly reduce heat loss from the surgical patient. The final method of preventing hypothermia, warming of fluids and blood

products, is essential in major surgical cases and trauma.

Heat exchange and fluid flow in warming devices. Russell (1969) analyzed factors that determine the effectiveness of warming in fluid warmers. These determinants of heat exchange were flow rate, length and conductivity of the intravenous tubing, and temperature gradient between the heat source (i.e., warm water bath or heating block) and the intravenous fluid. Various devices used today incorporate different designs to achieve fluid warming. Current warmers use one of the following four basic designs (a) single-coil immersion heater; (b) single-channel, dry wall heater; (c) multichannel, countercurrent heat exchanger; and (d) single-channel, countercurrent heat exchanger (Flancbaum et al., (1989).

The oldest of these devices, the single-coil immersion heater, warms fluid by circulating it through a plastic coil immersed in a heated water bath with a temperature approximately equal to 37^0 C. Newer, but similarly designed, single-channel dry wall heater place tubing coils in direct contact with a heating block. The ability of these units to warm and infuse fluids rapidly is hampered by long, narrow plastic tubes with low heat-transfer capacity and high back-

pressure that limits flow rates to 300 ml/min (Cherry, Hodgson & Nottebrock, 1981).

Due to these limitations, more efficient fluid warmers were developed. The multichannel, countercurrent heat exchanger and the single-channel, countercurrent heat exchanger (i.e., Level 1™ Fluid Warmer) are examples of this recent development in fluid warmer technology. Both warmers employ more conductive aluminum tubes (1,000-fold greater conductivity than plastic) interfaced with a countercurrent heated water bath. The Level 1™ Fluid Warmer uses a single large aluminum tube, while the multi-channel incorporates numerous small aluminum tubes. These designs maximize temperature gradients and heat transfer with the infused fluids (Flancbaum et al., 1989). The superior capability of these countercurrent heat exchanges allows them to warm and infuse intravenous fluids between a maximum rate of 1,200 ml/min (multichannel) and 1,800 ml/min (single-channel) (Flancbaum et al., 1989).

Besides improving the temperature gradient by using alunimum tubes, new fluid warming systems (i.e., Level 1™ Fluid Warmer) increase flow rate by using larger bore intravenous tubing. Enhancement of fluid delivery results from facilitating laminar flow, a

basic fluid dynamic property. In a hollow tube, streams of fluid running parallel to each other with little mixing is known as laminar flow (Barker & Tremper, 1989). With this smooth flow, fluid nearest the center of the tube travels faster than the fluid nearest the tube wall. Therefore, fluid velocity can increase without undue hinderance caused by friction against the wall.

Fluid delivery and intravenous catheter size. Intravenous fluid warmers are capable of reducing the incidence of hypothermia in surgical patients requiring large volumes of fluid. However, many of the warmers currently in use have both inefficient heat transfer and resistance to flow that limits the quantity of fluid they are able to infuse (Kruskall, Pacini, Ryan & Sutton, 1989). A newly developed fluid warmer (i.e., Level 1™ Fluid Warmer) is capable of heating and transfusing fluids faster and more efficiently than most conventional warming devices (Smith & Snider, 1989).

The Level 1™ Fluid Warmer's ability to deliver more fluid is partially the result of a larger bore output tubing which accommodates greater amounts of intravenous fluids (Smith & Snider, 1989). Due to its greater capacity, this tubing may also cause the fluid

to cool faster since more of the fluid is exposed to the cold operating room environment prior to entering the patient. At slower infusion rates, the risk of fluid cooling is obviously greater.

Decreased infusion rates can also result from using small intravenous catheters (Dula, Muller & Donovan, 1981). For example, an 18 ga catheter delivers two to three times less the amount of fluid than an 8.5 Fr catheter is able to infuse (Flancbaum et al., 1989). Because the Level 1™ Fluid Warmer is specifically designed for fast fluid delivery, its performance may be compromised if less than optimal size catheters are used.

Summary

Hypothermia is a body temperature equal to or less than 35^{0} C. Many factors contribute to the development of this condition and numerous problems can result. Therefore, prevention of the hypothermic state is of primary concern for the anesthetist.

An important maintenance measure is the warming of intravenous fluids. Numerous heating devices exist for this purpose, but many warm inefficiently and infuse too slowly. However, recent technological advances in fluid therapy have eliminated these inadequacies.

One of the newest technical innovations is the rapid heating fluid warmer. The Level 1™ Fluid Warmer is such an appliance and is capable of heating 1,000 ml/min of intravenous fluid. In order to derive the maximum benefit from this heating unit, a fast infusion rate is essential. With a less than adequate infusion time, as might occur with a small intravenous catheter, the efficacy of the Level 1™ Fluid Warmer may be compromised.

Chapter Two

Review of Literature

Boyan and Howland (1961) were the first to study and report the effects of cold and warmed blood transfusions in surgical patients. Initially, Boyan and Howland observed the physiological changes that occurred in patients receiving large quantities of cold blood. The most notable changes were seen in cardiac function. As body temperature decreased with the amount of blood transfused, numerous arrhythmias developed. When body temperature dropped to 27.5^0 C, cardiac arrest occurred. Along with these cardiac disturbances, Boyan and Howland observed shivering, mottled skin and extreme vasoconstriction in hypothermic patients.

Hoping to prevent these abnormalities, Boyan and Howland (1961) infused warmed blood in surgical patients. They developed a blood warmer for this purpose consisting of a 20 liter water bath maintained at 37^0 C, in which 24 feet of plastic tubing was immersed. At infusion rates of 50 ml/min to 100 ml/min, warmed blood temperature varied from 35^0 to

33^0 C. When rates increased to 120 to 150 ml/min, the temperature ranged between 32^0 and 30.6^0 C.

Boyan and Howland (1961) evaluated the warmer by transfusing between 7,800 ml and 9,600 ml of warmed blood into three surgical patients. Flow rates were as high as 60 ml/min for most of the transfusions. The patients' esophageal temperatures varied between 35.7^0 C to 36.7^0 C. All patients remained warm and pink, and their electrocardiograms were unchanged.

Boyan and Howland (1961) clearly demonstrated the benefits of warming blood prior to transfusion. However, many aspects of their methodology were lacking. They failed to report the total number of patients in their study and the general condition of these subjects at the time of the surgery. They also omitted describing the type of surgery performed and length of the procedures. No mention was made of controlling room temperature or other techniques used to prevent hypothermia. Regardless of these oversights, Boyan and Howland had discovered an important cause, and a subsequent prevention of hypothermia.

Later, Boyan (1964) reported the results of a study comparing two groups of patients, one receiving cold blood and the other receiving warmed blood.

Patients in each group underwent radical surgery for cancer, and each received at least 3,000 ml of blood at a rate of 50 ml or more per minute. The incidence of cardiac arrest in the cold blood group was related to the speed of transfusion. When 3,000 ml of cold blood was administered at a rate of 50 to 100 ml/min, 12 cardiac arrests were reported among 25 patients (48%). With transfusion amounts greater than 6,000 ml at a rate of more than 100 ml/min, the number of cardiac arrests was nine in eleven patients (81%). The total incidence of cardiac arrest in the cold blood group was 58.3%.

Patients in Boyan's (1964) warmed blood group were given 3,000 ml or more of blood at a rate equal to or greater than 50 ml/min. The same warmer described in Boyan and Howland's (1961) study was used. Out of the 118 patients in the warmed blood groups, eight cardiac arrests were observed, representing an incidence of 6.8%. The incidence of cardiac arrest in the cold blood group (21 of 36) and the warmed blood group (8 of 118) was statistically significant ($p < .01$).

Although Boyan (1964) had again proven the importance of warming blood, some of his methodology was unreported. As in his previous study with Howland (1961), the patients' general condition and length of

their surgery were omitted. Any attempt to prevent hypothermia by other means was also not reported. Nevertheless, the results of Boyan's study far outweighed the possible oversights in his reporting and research design.

These early studies by both Boyan and Howland (1961) and Boyan (1964) were limited to warming blood for administration. As more became known about hemorrhagic shock, other fluids gained importance as replacements for blood loss. Baue, Tragus, Wolfson, Cary and Parkins (1967) studied the use of Ringer's lactate solution to treat massive hemorrhage. Their *in vivo* research involved 18 canines that were bled at a rate of 5 ml per kilogram/min to a mean arterial pressure of 30 millimeters of mercury (mm Hg). The dogs were divided into three groups of six. Group one received only Ringer's lactate solution for their blood loss. The amount given was equal to four times the volume of red cells lost and 1.3 times the plasma contained in the shed blood. Group two received the same amount of Ringer's lactate solution determined for the first group; in addition, group two was also reinfused with one-half of their shed blood. Group three received only their shed blood as replacement. By means of a pulmonary arterial catheter, the status

of the dogs was evaluated by measuring and recording the following data: (a) pulmonary artery pressure, (b) pH, PO_2, and PCO_2 of arterial and mixed venous blood samples, (c) blood lactate, and (d) microhematocrits of all blood samples.

The study by Baue et al. (1967) confirmed that vascular volume restoration by a non-specific fluid without red cells satisfactorily increased cardiac output, improved oxidative metabolism, and corrected metabolic defects of hemorrhagic shock. Even when 73% of the dogs' blood volumes were replaced with Ringer's lactate solution, no deleterious effects occurred, and all animals survived. However, the authors suggested that Ringer's lactate solution alone may be unsuitable for massive hemorrhage in humans and that it should be supplemented with blood.

The researchers in the study ignored both hypothermia and fluid warming. Fortunately, similar research by Copping, Mather and Winkler (1972) dealt directly with the effect of fluid temperature. These authors studied the effect on dogs exsanguinated until a mean arterial blood pressure of 30 to 40 mm Hg was reached. Again the dogs were divided into three groups, yet all received Ringer's lactate solution. The amount administered was equal to four times the red

cell mass lost plus 1.3 times the plasma volume removed. The difference between the groups were the temperatures at which the fluid was intravenously infused.

The first group received Ringer's lactate solution at 4^0 C, the temperature at which "banked" blood was maintained. The second group of animals received the solution at room temperature (21^0-26.6^0 C). Group three received the Ringer's lactate solution warmed to 37^0 C via a blood warming coil immersed in water heated by a fluid warmer (Hemotherm^{TM}). All fluids were infused at a flow rate of 50 ml/min. Determinations of arterial blood pressure, pulse, and cardiac rhythms were made at base line, end-shock, and during infusion.

Findings of this study were most dramatic for the first group (i.e., those receiving cold fluids). Three of the five dogs in this group died of severe cardiac conduction defects during infusion of the Ringer's lactate solution. The other two dogs in this group displayed rhythm abnormalities, but survived. Rhythm disturbances were not seen in the animals receiving room temperature or warmed fluids. All dogs receiving cold fluids also had slowing pulses and lowered blood pressures. Conversely, infusions in group two and three produced significantly improved blood pressures

and little change in pulse rate. Esophageal temperatures decreased in all animals used in this study. While group one had an average of 3.4^0 C drop in their esophageal temperature readings, animals receiving room temeprature fluids displayed an average decrease of 2.3^0C. A slight 0.4^0 C mean temperature reduction occurred in the dogs administered warmed fluids.

The major oversight of this study by Copping, et al. (1972) was the failure of the researchers to record fluid temperatures at specific locations throughout the infusion period. They also neglected to measure room temperatures which may have caused considerable heat loss from both the animals and warmed fluids. Finally, they did not specify the length of the intravenous tubing from the warmer to the animal. The lack of this data makes the results of this study somewhat disputable.

The importance of the data excluded from the above in vivo study was identified and discussed thoroughly by Russell (1969). His observations were made while attempting to design a more efficient system for warming fluids. Russell recognized that heat loss occurs in the output line of the fluid warmer due to certain factors including (a) flow rate, (b) length of

the tubing, (c) conductivity of the layers, and (d) temperature gradient.

Russell (1969) observed that with a constant heat gradient (i.e., temperature difference between warmed fluid and cooler air), heat loss increased as flow rate declined. However, he found that the heat gradient was not constant, but decreased with greater flow. This reduction was due to less temperature rise in the heat exchanger (warm water bath) and also because of fluid cooling in the line. Russell suggested warming the environment to minimize this gradient effect and reduce heat loss.

Russell (1969) also advised using the shortest length of intravenous tubing possible. Not only does this reduce exposure of fluid to cool ambient temperatures, shorter tubing also has less resistance to flow. The opposite effect, greater resistance to flow, results from the use of male and female luer fittings commonly employed when adding extension tubing. For this reason, Russell believed in using only the tubing supplied in the warmer set.

The final factors discussed by Russell (1969) were the conductivity of the layers--fluid, plastic, and air. Heat was conducted from the fluid and then through the plastic tubing wall to the surrounding air.

Since the ambient temperature was usually markedly lower than that of the fluid in the tubing, circulating air removed heat by convection. Hence, with a larger diameter or longer length tubing, more fluid exposure occured, resulting in greater heat loss. Increasing the thickness of the plastic tubing did little to prevent this loss. However, Russell (1969) suggested that insulating the tubing or increasing the flow rate would benefit heat retention.

Faster flow rates are possible with larger diameter intravenous catheters. Dula, Muller & Donovan (1981) studied the fluid flow rate capabilities of commonly used intravenous catheters using different infusion methods. After determining that flow rates for Ringer's lactate solution were identical to that of tap water, intravenous plastic bags of tap water were infused by gravity and pressurized flows through various sizes of intravenous catheters. Gravitational flow was achieved by hanging the fluid bag at a height of 1 meter (100 centimeters [cm]) above the catheter. The pressurized flow was maintained by a pressure cuff wrapped around the fluid bag with a constant pressure of 200 mm Hg maintained during the infusions.

Dula et al. (1981) infused the tap water through various gauges of intravenous catheters that included

(a) 18 ga, (b) 16 ga, and (c) 14 ga. All catheters were 2 inches long. Flow rates were then determined by infusing the tap water via gravity and pressure through the different catheters. Rate calculations were made for each 100 ml increment. A total of three incremental infusions were done for each catheter to insure accuracy.

Results of the study by Dula et al. (1981) showed that both pressure and larger diameter catheters increased flow rates. For example, the 14 ga catheter was able to infuse 180 ml/min by gravity and 330 ml/min with pressure. The 18 ga catheter produced the slowest gravitational and pressurized flow rates with measurements of 60 ml/min and 200 ml/min respectively.

Dula et al. (1981) identified important factors to consider when determining intravenous flow rates. However, an 8.5 Fr catheter was not included in their study. Because of the 8.5 Fr catheter's wide usage, its omission detracts from Dula et al.'s otherwise thorough study of intravenous flow rates.

Baker (1985) investigated the correlation of flow rate with heat loss in warmed intravenous fluids using a conventional coil water bath type warmer. He discovered an apparent net loss of temperature for fluid infusing at rates below 400 to 500 ml/hour.

Baker attributed this heat loss to convection by cool ambient air. At flow rates above 3,000 ml/hour, fluid temperature in the warmer outlet tubing also decreased. However, this decrease in temperature resulted from exceeding the fluid warmer's capacity. Therefore, warmer technology at the time of this study limited flow rates to 3,000 ml/hour for maximum efficiency.

Recent technological advances in fluid warmer design allow efficient heating at much greater flow rates. Fried, Satiani and Zeeb (1986) developed and tested a Rapid Solution Administration Set (RSAS). They incorporated an extracorporeal heat exchanger, in combination with (a) an intravenous spike, (b) a 40-micron screen blood transfusion filter, (c) a fluid shutoff valve, (d) two 170-micron filtered drip chambers, (e) a male luer perfusion adapter, (f) 1/4-inch internal diameter polyvinyl chloride tubing, and (g) a macrodrip intravenous administration set. A 20 liter per minute counter-current water flow provided the heat exchange medium across the extracorporeal heat exchanger for warming of intravenous fluids. This counter-current flow runs through aluminum tubes in the heat exchanger in an opposite direction of the fluids being infused. The fluids are warmed via conduction through the special coated aluminum which prevents

direct fluid contact with the metal surface. Water temperature in this system was maintained at 40^0 C.

Fried, et al. (1986) performed both _in vivo_ and _in vitro_ testing of the new warmer. In the _in vitro_ portion of the study, infusions of plasmalyte-A solution and packed human red blood cells (PHRBC) were used. The center of the solution bags were 91 cm above the 8.5 Fr catheter located at the distal end of the system. Using a pressure infuser (Fenwal Laboratory, Deerfield, IL) various pressures (0-300 mm Hg) were exerted on the solution bags. Gravitational and pressurized flow rates were measured by the use of a stopwatch and a graduated cylinder. The infusion catheter was positioned at the top of this collection cylinder. Fluid temperature was determined by temperature probes located at the inlet and outlet ports of the extracorporeal heat exchanger.

The _in vitro_ portion of the study by Fried et al. (1986) found that at a constant flow of 500 ml/min and an inlet baseline temperature of 4^0 C, plasmalyte-A solution reached a steady temperature of 38^0 C in one minute. Red blood cells achieved a slightly higher temperature of 38.4^0 C at the outlet port, but the inlet baseline reading was also considerably elevated at 14^0 C. Infusion flow rates increased similarly for

both infusing solutions with pressures up to 300 mm Hg. Hence, the authors concluded that the usual viscosity variance in flow between PHRBC and crystalloids is negligible when large-bore tubing and catheters are used.

In their in vivo study, Fried et al. (1986) used an anesthetized canine. The dog's left femoral artery was cannulated with a 12 ga catheter for controlled blood removal. The blood was placed in heparinized transfusion bags. The right femoral vein was cannulated with an 8.5 Fr catheter and connected to the RSAS for return of the exsanguinated blood. After removing 1,200 ml of whole blood, the animal's blood was warmed and re-infused. Inlet and outlet infusate temperatures and infusion flow rates were measured and recorded. Hemodynamic parameters were also noted during and after the infusion. When the dog was hemodynamically stable, another 1,200 ml of blood was exsanguinated and replaced with 4-day-old compatible canine packed red blood cells (CPRBC). The temperature of the CPRBC was 10^0 C.

Results of the in vivo study substantiated the effectiveness of the RSAS. At flow rates varying from 710 ml/min to 730 ml/min, both the collected blood and CPRBC were warmed to 38^0 C just before entrance into

the animal's vasculature. The dog's core temperature did not drop below 35^0 C throughout the study, and hemodynamic stability was maintained, as determined by cardiac outputs, hemodynamic pressures, pulmonary artery temperature, and laboratory analyses of blood samples.

The findings of both the in vivo and in vitro study by Fried et al. (1986) clearly demonstrated the efficiency of this newly devised fluid warming system. However, in reporting their findings, the researchers failed to mention room temperature, which may have affected fluid and body temperature. Even without this data, the investigators presented sufficient evidence to warrant further investigation of the RSAS.

Santiani, Fried, Zeeb and Falcone (1987) again evaluated the performance of RSAS in treatment of 33 consecutive multiple-trauma patients with severe hypovolemic shock. The patients ranged in age from 16 to 65 years, with an average of 32 years. While penetration injuries occurred in 10 of the victims, blunt injury from motor vehicle accidents was the primary cause of trauma.

All patients received a massive amount of fluids warmed by the RSAS through an 8.5 Fr vein catheter. Varying amounts of crystalloid, colloid, and blood

products were administered to these patients, and flow rates were individualized. An average of 19 units of packed red blood cells, 10 units of fresh-frozen plasma, as well as other colloids, and 12 liters of crystalloid were given to each patient in the initial 24 hours of hospitalization. The majority of these fluids were administered in the first 2 hours of treatment.

The study proved the RSAS reliably provided outflow fluid temperatures of 37 to 38^0 C regardless of the inflow fluid temperature. Initial post-resuscitation body core patient temperatures were maintained at an average of 35.2^0 C. Although these temperatures appear to reflect favorably on the performance of the RSAS, firm conclusions are not possible due to lack of important related data. Santiani et al. (1987) neglected to report environmental temperatures or any other means utilized to prevent hypothermia in these trauma victims. They also failed to measure or standardize the length of the output tubing from the warmer to the patient. The fact that various infusion rates were used also complicates interpretation of the findings. Thus, while generalizations were made about the efficacy of the RSAS, this research provided little concrete evidence

about its performance.

Another study involving the RSAS, also known as the Infuser 37, was done by Flanchbaum, Trooskin & Pedersen (1989). They formulated a theoretical model for the design and operation of fluid warmers. The authors also described useful "apparent thermal clearance" which measures the intrinsic efficiency of these devices. Experimentally, apparent thermal clearance can be derived by solving an equation. Physically, this factor is the rate of flow at which the temperature rises to 63% of its limiting value.

The RSAS was compared to a similar warmer, the Level 1™ Fluid Warmer. Two major differences between these warmers are their priming volumes and their heat exchangers. The RSAS requires 125 ml of fluid for priming, while the Level 1 needs only 65 ml. Both of these warmers use heated water in a countercurrent heat exchanger for warming fluids, but the Level 1 uses a single-channel (i.e., one aluminum conduit) versus the multichannel of the RSAS.

Flanchbaum et al. (1989) tested each of the warmers with normal saline and undiluted packed red blood cells. Inflow temperatures were between 2 and 10^0 C. Outflow temperatures were measured by a digitalized thermistor located 25 cm from the heat

exchanger. Flow rates were varied using a peristaltic pump capable of infusing water at 2 liters/min. The apparent thermal clearance was then calculated for each fluid warmer.

Flanchbaum et al. (1989) theoretically determined that an apparent thermal clearance of at least 400 ml/min is needed for a blood-warming device to be clinically suitable as part of a rapid infusion system. Only the Level 1™ Fluid Warmer met this rigorous standard, surpassing the minimal acceptable thermal clearance by 258 ml/min. Additionally, the apparent thermal clearance for saline was quite impressive at 1,356 ml/min. The authors concluded that the Level 1™ Fluid Warmer's single channel, countercurrent heat exchange unit was the only one efficient enough to meet their proposed criteria.

While properly demonstrating the Level 1™ Fluid Warmer's superior capabilities over that of the RSAS, the researchers did not report room temperature, which may have contributed to either heat loss or gain. This information was particularly pertinent to this study, since initial outflow temperature was recorded 25 cm from the warmer instead of at the warmer's outlet. Previous studies have revealed the actual and potential heat loss in warmer output tubing (Russell, 1969).

Subsequently, all relevant factors affecting the performance of these rapid influsion warmers were not considered in this study.

Summary

Many early studies recognized the untoward physiological ramifications resulting from infusions of cold intravenous solutions. These detrimental effects are often seen in surgical patients requiring large amounts of fluids rapidly to support hemodynamics. Warming these fluids is essential in preventing hypothermia and its related side effects. Therefore, in order to administer large quantities of warmed fluids rapidly, an efficient fluid warming device must be used. The Level 1™ Fluid Warmer is capable of this task. However, studies of conventional fluid warmers have shown that significant heat loss can occur in the output tubing, especially at slower infusion rates. The use of small diameter infusion catheters can severely limit flow rates. Fluid cooling may even occur in output tubing from a rapid infusion warmer if inadequate diameter catheters are used. Therefore, a study was designed and implemented to determine if fluids, warmed by the Level 1™ Fluid Warmer, lose heat when infused through small diameter catheters.

Chapter Three

Methodology

Design

In order to discover whether the intravenous catheter size causes significant fluctuations in the temperature of Ringer's lactate solution warmed by the Level 1™ Fluid Warmer, a quasi-experimental design was used. This particular design was selected because the use of a control group and randomization were not possible. The four different sizes of catheters used in this study were (a) 18 ga, (b) 16 ga, (c) 14 ga, and (d) 8.5 Fr. Three 1,000 ml bags of Ringer's lactate solution were warmed and infused through each of the four catheter sizes. Fluid temperature was measured and compared at designated points throughout the infusion period. The major temperature comparison for this study was the difference in fluid temperature between the warmer outlet and the hub of the intravenous catheter.

Population and Sample

The population consisted of one Level 1™ Fluid

Warmer. The sample for the study was obtained from the main operating room at a mid-Atlantic, university teaching hospital.

Instrumentation

Level 1™ Fluid Warmer (model H-500). The Level 1™ Fluid Warmer is a rapid infusion fluid warming system using a single-channel countercurrent heat exchanger. This exchanger uses a conductive aluminum tube interfaced with a countercurrent heated water bath that maximizes temperature gradients and heat transfer with the infusate. The Level 1™ warms intravenous fluids from 10^0 C to a minimum of 35^0 C at flow rates up to 1,000 ml/min. The system consists of two components, a hardware system and a disposable set. The hardware system employs an electrical warmer and pump that warms the enclosed water supply to 40^0 C and circulates it through the heat exchanger. The disposable set consists of the following: (a) large bore bag spike and Y-set (3.8 mm ID), (b) high efficiency heat exchanger, (c) 170-micron gross blood filter, (d) automatic air eliminator, and (e) 6 foot patient line (3.3 mm ID). (Level 1 Technologies Incorporated, 83 Enterprise Drive, Marshfield, Massachusetts 02050).

Mon-A-Therm™ Model 6500 Temperature Monitoring System. The Mon-A-Therm™ Model 6500 two channel temperature monitoring system was used to measure fluid temperature at designated points. The system measures and displays temperatures from 1.0 to 50.0^{0} C with an accuracy of $\pm$ 0.1^{0} C. The system uses a pre-set calibration with an automatic check and is shielded against radio frequency interference. (Mon-A-Therm Incorporated, 520 South Jefferson Avenue, St. Louis, Missouri 63103).

Temperature probes. Four Mon-A-Therm™ luer lock temperature probes and one Mon-A-Therm™ skin temperature probe were used in this study. Along with sensing room temperature, the luer lock probes measured fluid temperature at the exit point of the warmer, the exit point of the air eliminator, and the end of the output tubing. The skin sensor monitored the temperature of the bags containing Ringer's lactate solution. (Catalog number 503-0501, Mon-A-Therm Incorporated, St. Louis, Missouri 63103.)

Intravenous catheters. Three Critikon intravenous catheters were placed at the end of the warmer's output tubing. These catheters ranged in the following sizes: (a) 18 ga, 1 3/4 inch, (b) 16 ga, 2 1/4 inch, and (c) 14 ga, 2 1/4 inch. (Catalog numbers 4424, 4458, and

4472, Critikon, Tampa, Florida 33607.) One Arrow 8.5 Fr, 4 inch catheter was also attached to the end of the warmer's output line. (Arrow Company, Reading, Pennsylvania 19610.)

Three Medex Hi-Flo™ 3-way stopcocks with male luer locks were used. One stopcock was connected at the exit of the warmer, another at the exit of the air eliminator, and the third between the end of the output tubing and the hub of the intravenous catheter. This allowed for the placement of the luer lock temperature probes. (Part number MX931-IL, Medex Incorporated, 3637 Lacon Road, Hilliard, Ohio 43026.)

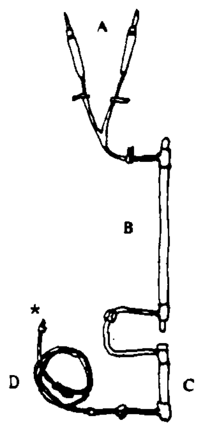
Procedures

The study was conducted at the medical center under simulated operating room conditions. Typical of an operating room at any major hospital, this room was set-up to replicate an actual surgical environment. All equipment and lights normally used were activated. The room temperature was maintained at 21.0 ± 0.5^{0} C. The Medex stopcocks used to connect the luer lock temperature probes were placed in the Level 1™ Fluid Warmer's disposable set at three points (a) the exit point of the fluid warmer, (b) the exit point of the air eliminator, and (c) the end of the output tubing.

The luer lock probes were then attached and connected to the temperature monitors. A skin temperature probe was placed on each bag of Ringer's lactate solution. Recorded temperatures of the fluid bags were 22.0 $\pm$ 0.5^0 C.

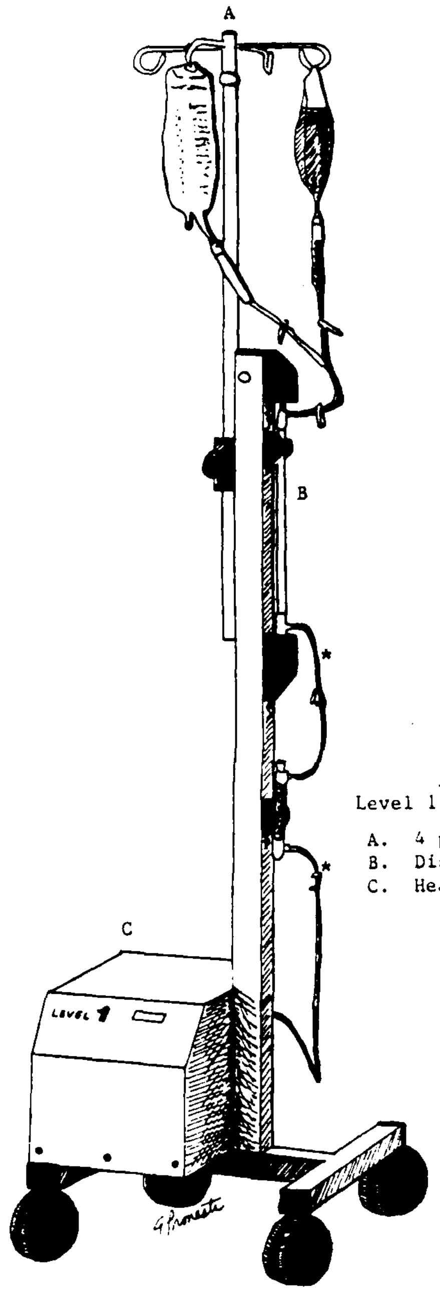
The Level 1™ Fluid Warmer and temperature monitoring points are depicted in Figure 1. The fluid warmer was turned on and allowed to reach its operating temperature of 40^0 C. The disposable tubing was then primed with 65 ml of Ringer's lactate solution for a 10 minute equilibration time period. The intravenous infusion catheter under investigation was then placed on an armboard attached to the operating room table. The end of the output tubing was connected to the intravenous catheter via a 3-way stopcock. Liter bags of Ringer's lactate solution were then hung on the Level 1™ Fluid Warmer's four-prong pole and connected to the disposable infusion set. The bottom of the bags were 108 cm above the infusion catheter.

When preparations were completed, 1,000 ml of Ringer's lactate solution was infused through the warmer system for each of the four different sizes of intravenous catheters. Fluid temperatures were recorded every 30 seconds for the first 5 minutes and every minute thereafter at each of the points specified



D-100 Disposable Set

A. Large bore bag spikes
B. Heat exchanger
C. Air eliminator
D. Large bore output tubing



Level 1™ Fluid Warmer

A. 4 prong IV pole
B. Disposable set
C. Heater and pump

Figure 1. Level 1™ Fluid Warmer and Disposable Set (D-100)

Note: Points of temperature measurements are indicated by *.

in Figure 1. A total of three 1,000 ml bags of Ringer's lactate solution were infused through the system for each intravenous catheter to insure accuracy.

Statistical Analysis

Statistical analysis for the effect of the intravenous catheter size on the temperature of fluids warmed by the Level 1™ Fluid Warmer was examined by a one-way analysis of variance or one-way ANOVA. Because temperatures basically stabilized after 3 minutes, this one-way ANOVA was used to interpret the data observed in the first 3 minutes of the infusion. Logically then, the parametric procedure tested the significance of differences between the mean temperatures of the fluid leaving the warmer and the fluid exiting the output tubing during this 3 minute time period.

Chapter Four

Results

The hypothesis was tested using a quasi-experimental design. The purpose of this study was to determine if fluids warmed by a rapid infusion warmer (i.e., Level 1™ Fluid Warmer) lost heat at flow rates limited by intravenous catheter size (see Table 1). The independent variable was intravenous catheter size. The dependent variable was the difference in fluid temperature from the exit point of the warmer to the entry point of the patient. The mean fluid temperatures recorded for each intravenous catheter along with temperature losses are listed in Table 2.

Temperature measurements during the first three minutes were examined by a one-way ANOVA. The statistic computed by the ANOVA, the F-ratio, identified the variance between the independent variable groups (see Table 3). A F-ratio of 225.691 was calculated for catheter size which corresponded to $p < .001$. Therefore, catheter size had a significant effect on fluid temperature.

Table 1. <u>Mean Flow Rate by Gravity* for Each Intravenous Catheter Size</u>

Mean flow rate (ml/min)	Catheter size
59	18 ga
122	16 ga
167	14 ga
333	8.5 Fr

*Fluid bag at a height of 108 cm

Table 2. <u>Intravenous Catheter Sizes with Mean Fluid Temperatures</u>

	Mean Temperatures (^{0} C)		
Catheter Size	Exit Point	Entry Point	Loss
18 ga	33.9	28.4	5.5
16 ga	33.1	28.7	4.4
14 ga	32.7	30.4	2.3
8.5 Fr	34.5	35.4	-0.9

Table 3. Results of the Analysis of Variance

Source	Sum of Sqs.	df	Mean Sq.	F-ratio	p
Catheter Size	429.489	3	143.163	225.691	<0.001

In conclusion, results of the multifactor ANOVA indicated that intravenous catheter size significantly affected heat loss from fluids warmed by the Level 1™ Fluid Warmer. Heat loss was greatest with the 18 ga catheter. In the case of the 8.5 Fr catheter, there was no heat loss. Conversely, fluid temperature continued to rise slightly after leaving the warmer. As catheter size decreased from the 8.5 Fr size, the heat loss progressively increased.

Chapter Five

Discussion

The purpose of the quasi-experimental study was to determine if fluids warmed by a rapid infusion warmer (i.e., Level 1™ Fluid Warmer) lost heat at flow rates limited by intravenous catheter size. Results of the study revealed that a significant heat loss occurred with smaller catheters (i.e., 18 ga, 16 ga, and 14 ga). Therefore, the hypothesis, which stated there was no difference in fluid temperature from a rapid infusion warmer at flow rates determined by varying intravenous catheter diameters, was rejected.

Correlation with Previous Studies

Results of this study suggested that heat loss occurred in the output tubing from a rapid infusion warmer when flow rates varied with intravenous catheter size. Slower flow rates resulted in greater heat loss. This finding supported observations made by Russell (1969) and Baker (1985) who identified heat loss in conventional warmer output tubing. Flow rates for the smaller catheters (i.e., 18 ga, 16 ga, and 14 ga) used

in this study differed from those reported by Dula et al., (1981). While there was only a slight 1 ml/min difference between the 18 ga catheter; rates for the 16 ga and 14 ga catheters exceeded those of Dula et al., (1981) by 22 ml/min and 42 ml/min respectively. The faster rates in this study were probably attributable to the greater height of the fluid bags (i.e., 108 cm vs 100 cm) increasing gravitational force. Also, the larger diameter tubing (i.e., 3.3 mm ID vs. 2.6 mm ID) used on the rapid infusion warmer's disposable set enhanced laminar flow resulting in increased fluid velocity.

Effect of Intravenous Catheter Size on Heat Loss

Statistical analysis demonstrated that intravenous catheter size had a significant effect on heat loss. A two-way analysis of variance for the fluid temperatures recorded during the first 3 minutes of infusion for the various catheters resulted in a $p < .001$. An inverse relationship existed between catheter size and heat loss. As the catheter size became smaller, heat loss increased. The reduced flow rates accompanying the smaller catheters probably allowed more heat loss to cool ambient air by convection. Mean temperature declined from 5.5^0 C for the 18 ga catheter to less

than zero (-0.9^0 C) for the 8.5 Fr. This "less than zero" temperature reduction meant that the fluid temperature actually increased after leaving the warmer. During the infusion through the 8.5 Fr catheter, fluid temperature rose 0.9^0 C (34.5^0 to 35.4^0 C) from the warmer to the hub of the catheter. No plausible explanation existed for this rise in temperature.

Difficulties With the Study

The only difficulty with this study was securing the use of the Level 1™ Fluid Warmer. The university hospital had only one such warmer, and its availability was limited.

Recommendations for Further Study

1. Pressure devices are frequently applied to fluid bags to increase flow rates. Replicate the study using intravenous fluid bags pressurized to 300 mm Hg.

2. Replicate the study using packed red blood cells instead of Ringer's lactate solution to identify effects of greater viscosity on fluid flow.

Conclusions

The hypothesis was rejected as statistical

analysis revealed a significant heat loss from fluids, warmed by a rapid infusion warmer, at flow rates limited by 3 of the 4 intravenous catheter sizes studied. Based on this data, the following conclusions were made: (a) loss of heat from intravenous fluids warmed by a rapid infusion warmer (i.e., Level 1™ Fluid Warmer) could be prevented by using an 8.5 Fr intravenous catheter with maximal gravitational flow; and (b) as intravenous catheter diameters decreased below the 8.5 Fr size, progressively greater heat loss occurred from fluids warmed by the rapid infusion warmer.

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VITA

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